Comments of the California Trucking Association on Prioritization of Toxic Air Contaminants Under the Children's Environmental Health Protection Act.

Comment 1: OEHHA failed to follow the intent of SB 25 (Escutia, 1999) which requires identification and prioritization of chemicals that have a disproportionate impact upon infants and children

SB 25 enacted the Children's Environmental Health Protection Act, which requires CARB to review criteria pollutant standards and toxic control measures to protect the health of infants and children. Once reviewed, CARB is to determine if current standards are health protective, considering increased susceptibility of infants and children to environmental contaminants.

OEHHA is required, by July 1, 2001 to establish a list of up to five toxic air contaminants (TACs) to which infants and children would be exposed that result in a disproportionately high exposure and/or exhibit special susceptibility.

The criteria set in SB 25 to determine which chemicals may cause special susceptibility to illness are:

1) Exposure patterns among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population,
2) Special susceptibility of infants and children to ambient air pollutants in comparison to the general population,
3) The effects on infants and children of exposure to toxic air contaminants and other substances that have a common mechanism of toxicity and
4) The interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants.

The focus is twofold; OEHHA is to look at disproportionate exposure and the disproportionate susceptibility to disease in infants and children. The method of prioritization utilized in the Draft Report focuses on prioritizing the list of TACS with respect to the general population and statewide exposure. This general ranking does not accomplish the intent or stated criteria established in SB 25.

A logical method to prioritize the disproportionate risk to infants and children would be to start with the diseases to which infants and children have a higher or disproportionate incidence of risk such as leukemia, Hodgkin's lymphoma, central nervous system cancer and brain cancer. Once the diseases have been identified, a focused literature review evaluating the chemicals that are scientifically linked to these childhood diseases would determine which chemicals are suspected of causing disproportionately high risk to children and infants.

Recommendation 1: Focus on childhood diseases with disproportionate incidence of risk and rank those chemicals by exposure.

Response: OEHHA believes that the approach used does address the requirements of the statute. We evaluated information pertinent to the issues of both exposure and susceptibility to toxicant effects.

The comment is incorrect in suggesting that "the Draft Report focuses on prioritizing the list of TACS with respect to the general population and statewide exposure". General population exposure and toxicity were properly considered as part of the available data, but were not the exclusive input into the prioritization. The law requires OEHHA to consider general population exposures when prioritizing the TACs under SB 25. In particular, ambient concentration data and emissions inventory information as well as data on the known toxicity of TACs were used in the early stages of screening to determine which compounds might be relevant, in terms of endpoint and/or exposures in California, for further, more detailed consideration. The use of general toxicity and

exposure data was necessitated by the fact that for many of the less well-studied TACs data specific to infants and children or in immature experimental animals were not available to OEHHA. In addition to this prioritization using general toxicity data, OEHHA specifically considered evidence of differential toxicity to infants and children in the few cases where these data were available. This process appears to be at least broadly consistent with the recommendation by the commenter, in that having assembled these general toxicological data, OEHHA was specifically interested in toxicological endpoints which might reasonably be expected to have differential impacts on children. (Further explanation of the use of general toxicity and exposure data in the initial stages of prioritization is given in the response to comments 1 - 4 from the Chemical Industry Council of California, on pages 1-3 of those responses.)

Comment 2. OEHHA's ranking of chemicals is suspicious, unclear and not fully explained in the Draft Report - hindering CTA's submittal of meaningful comments.

The criteria used by OEHHA to prioritize the list of current TACs is unclear and not well documented in the Draft Report. OEHHA began with the list of 200 TACs and ranked them by toxicity and exposure. Special impacts to infants and children were not considered. Rather than focusing on unique exposures to children based on their general environment such as indoor floor level exposure (average household) or outdoor environments including soil and grass, OEHHA focused on the ratio of general chronic Reference Exposure Levels (RELs) to ambient air concentration, producing a group based on a general ratio of highest non-cancer toxicity combined with highest exposure. A second group was created using the ratio of carcinogens (using the cancer unit risk factor) and ambient air concentrations. The two groups of rankings were then combined.

The combination of these two groups is vague. The Draft Report inadequately explains the scientific method utilized by OEHHA. It appears by the limited information presented in the Draft Report that the two groups are apples and oranges and should not

be combined to represent risk to the general public, and especially not the disproportional risk to infants and children.

Recommendation 2: Provide CTA with all chemicals and their numeric ranking in the first two groups as well as the combined group to allow meaningful comment.

Response: OEHHA regrets any lack of clarity in the Draft report, and will endeavor to resolve this, either in these responses or in revisions that may be made, in the light of these and other comments, to the final document. In the draft document, OEHHA explained the steps used in the prioritization

The process used for the initial prioritization using chronic RELs and potency factors, along with exposure data has already been addressed in the response to comment 1, and is explained in the draft report. The process for combining the list of carcinogens and non-carcinogens is explained in the OEHHA draft document, paragraph 4 of section IIA in the Introduction (page 3-4). This process necessarily involved an element of scientific judgment, and OEHHA sympathizes with the comment's view that the two types of toxic endpoint are "apples and oranges" which are difficult to compare (although note should be taken of recent efforts to facilitate such comparisons, as described in the US EPA's recent drafts of the revised Carcinogen Risk Assessment Guidelines and in some recent carcinogen risk assessments by OEHHA). However, the wording of the legislation specifies a single list and therefore requires OEHHA to make such a comparison by the best available means.

With regard to recommendation 2, unfortunately the timetable imposed on OEHHA, the Scientific Review Panel for Toxic Air Contaminants and the Air Resources Board by the legislative mandate does not allow further opportunities for public comment at the present stage. However OEHHA is providing more information in the Appendix concerning the prioritization process.

Comment 3: The diesel particulate unit risk factor is based on occupational exposures, does not reflect today's diesel fuel or today's engines, and is a poor indicator of exposure to infants and children.

The "quantitative risk assessment" ("QRA") prepared by OEHHA and a "unit risk factor" ("URF") selected by the SRP were based upon the OEHHA QRA. As CARB admits, however, OEHHA's QRA and the SRP's URF are based on "exposures to emissions from historical diesel fuel formulations and engine technologies." (CARB Resolution 98-35.)

OEHHA's QRA and the SRP's URF are based on a study by Dr. Eric Garshick of lung cancers in railroad workers assumed to have been exposed to unknown levels of diesel locomotive exhaust between 1959 and 1980. Despite the lack of exposure information and the known differences in particulate emissions from modern truck engines and old locomotive engines, this study is still being used as a basis for OEHHA's QRA.

The scientific community and Dr. Eric Garshick himself have declared the railroad workers study is not usable for QRA even as to historical diesel locomotive exhaust. Among the scientific authorities that CARB rejected in relying on the Garshick railroad workers study as the basis for the Diesel TAC Listing were the following:

- Dr. Eric Garshick, the author of the railroad workers study, who testified that his work is not usable for quantitative risk assessment and did not support OEHHA's QRA.
- Dr. Kenny Crump, retained by the United States Environmental Protection Agency ("USEPA"), who independently reviewed the railroad workers data and concluded that, because workers believed to have been exposed to diesel locomotive exhaust longer had fewer lung cancers than those believed to have been exposed less, the data did not support OEHHA's QRA.

- The Health Effects Institute ("HEI") -- an independent scientific body partially funded by the USEPA-- which convened an independent panel of preeminent

scientists to review the adequacy of the railroad workers study for a QRA. The HEI "Diesel Epidemiology Expert Panel" (the "HEI Diesel Expert Panel") released a report in June 1999 which both questioned the reliance on estimated exposures to "historical" diesel exhaust and found that the Garshick railroad workers study is not usable for a QRA for the same reason identified by Dr. Kenny Crump.

- The federal Clean Air Science Advisory Committee ("CASAC") -- an independent, "blue ribbon" panel of scientists appointed to advise the USEPA-- issued an October 1998 report rejecting much of the science relied upon by OEHHA. Significantly, CASAC noted the "substantial differences between emissions from engines produced since the early 1990s and those to which human and animal subjects comprising our present health database were exposed…" One member of CASAC expressly warned the USEPA not to rely upon the "mathematical gymnastics" utilized by OEHHA.

- The latest version of the USEPA diesel document states that a number or range cannot be estimated from the studies available today and suggests diesel a likely carcinogen.

The reports issued by the HEI Diesel Expert Panel and CASAC, as well as the testimony and reports of other preeminent scientists, demonstrate that the best available science, and the only science (the railroad workers study) on which OEHHA and the SRP relied for the QRA, does not support the Diesel TAC listing. CARB's summary rejection of these reports, which comprise the best and brightest independent science that exists on the relevant topic, demonstrate that CARB's Diesel TAC listing was an arbitrary political decision based neither on the "best available scientific evidence" nor on "sound scientific knowledge."

CARB's "risk characterizations" for hypothetical highways, truck stops and distribution centers are overestimated based on actual measurements. Using the SRP URF (despite its

rejection by the scientific community) and purposefully unrealistic exposure scenarios (including the assumption that infants and children are exposed to the same diesel exhaust source 24 hours per day, 365 days per year, for a 70 year lifetime [*i.e.* 25,550 days in a row], without alteration in wind direction, receptor location, or even time off for indoor sleep and play), CARB is forecasting significant health risks throughout California for infants and children despite an absence of scientific support or medical evidence of the diesel-driven lung cancer "epidemic" predicted by CARB's models.

CARB's and others' predictions all ultimately rest upon OEHHA's scientifically indefensible extrapolations from unknown exposures to historical diesel locomotive exhaust.

Recommendation 3: Discontinue using the diesel particulate unit risk number that is based on occupational exposures that are unrelated to children and infants.

Response: Virtually all of the bulleted concerns raised in this comment were thoroughly debated during the identification phase for diesel exhaust as a Toxic Air Contaminant and will not be debated here. For more information, the reader is referred to the document: *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant, Appendix III, Part C Responses to Comments (ARB, 1998b).*

The validity and applicability of the diesel exhaust cancer unit risk factor (URF) have been thoroughly documented in the diesel exhaust Toxic Air Contaminant (TAC) document. That document includes a section on the uncertainties inherent in extrapolating from the quantitative cancer risks associated with occupational diesel exhaust exposure to the quantitative cancer risks associated with ambient exposure. The comment indicates that CARB has conducted a quantitative risk assessment for infants and children of lung cancer from diesel exhaust exposure. In fact, this is not the case. This draft OEHHA document is not a risk assessment; rather it is a hazard identification document.

The prioritization summary for benzo[*a*]pyrene and other polycyclic aromatic hydrocarbons (PAHs) describes data indicating that infants and children are more sensitive than adults to the carcinogenic effects of PAHs. Since diesel exhaust contains a variety of PAHs, the diesel exhaust URF, which is based on adult occupational exposures, may underestimate the cancer risk to infants and children. Note also that we are concerned about noncancer health impacts of diesel exhaust in evaluating diesel exhaust particulate matter for listing under SB 25, not just the carcinogenic properties.

Comment 4: OEHHA has failed to distinguish diesel particulate exposure from overall particulate exposure and is simply singling out diesel particulate.

The risk from diesel particulate is not different than the risk from every other combustion source particulate. Diesel PM cannot be measured directly and is often estimated from elemental carbon (EC) measurements. Distinguishing ambient diesel particulate concentrations from overall particulate concentrations is impossible and certainly does not provide an accurate picture of vehicle emissions in today's environment or exposure to infants and children.

Recent published emissions data from in-use light, medium and heavy duty diesel vehicles have shown that the elemental and organic fractions are highly variable and a function of driving cycle, fuel type, technology, sampling method and overall particulate emission rate.

Durbin et al. (1998) tested 15 MD/LHD in-use diesel vehicles using California Diesel fuel over the FTP. The overall average percentage of elemental carbon was 64.0% +/- 21% with a high of 90.5% and a low of 28.9%. There was an overall trend that the higher emission vehicles tended to have higher percentages of organic carbon. The emission rate of these vehicles varied from 57.9 mg/mile to 767.7 mg/mile.

Schauer et al. (1999) tested two medium diesel trucks on California diesel fuel over the hot FTP cycle and found that elemental carbon was 30.8%. The percentage of organic matter depended highly on the sampling method used and varied from 19.7% to 30.4%. Lowenthal et al. (1994) reported emission characterization of heavy-duty diesel vehicles. They tested a relatively large fleet of buses and trucks and found that the elemental fraction varied from 5% to 61%. The vehicles with the lowest percentage of elemental carbon were urban buses equipped with particulate traps.

The Desert Research Institute (Gillies and Gertler, in press) evaluated studies of mobile source particulate profiles and speciation. They report that there is wide variation in the ratios of elemental and organic fraction particulates in both gasoline and diesel vehicles, and that the contribution of gasoline vehicles to elemental carbon emissions is much larger than previously believed.

Summarizing results of three studies including EPA's SPECIATE database, CE-CERT's and NFRAQS, DRI noted that the Elemental Carbon to Organic Carbon (EC/OC) ratio in diesel-fueled vehicles ranged from 0.48 to 2.79. For gasoline engines, the EC/OC ratio varies widely with some studies showing variation from 0.02 to 17.0. The report concludes that the relative amounts of these two components in particulate emissions from both diesel and gasoline vehicles is highly variable and includes considerable overlap. Therefore, attempting to apportion gasoline and diesel PM emissions on the basis of EC or OC fractions is highly suspect.

Recent studies by Christoforou et al. (2000) indicate that EC concentrations in the atmosphere are decreasing in Southern California and OC concentrations are increasing. The reasons for this are unclear but may be the result of decreases in gasoline-powered vehicles; decreases in diesel powered vehicles, or both.

There are numerous sources of elemental carbon other than mobile sources. These include other combustion sources such as cooking, wood burning stoves and fireplaces,

and wildfires. These factors have not been incorporated, nor accounted for, in the estimate.

There is now a substantial database of information demonstrating that estimating diesel particulate emissions from measured elemental carbon data is incorrect and must be re-evaluated.

Recommendation 4: Eliminate diesel particulate exposure from the Tier 2 list, as it can not easily be distinguished from overall particulate exposure.

Response: Some combustion source particulates may well be qualitatively similar in composition (e.g. PAH content). However, OEHHA is unaware of any published quantitative risk assessment indicating that a cancer risk exists for all combustion source particulates, and that risk is equivalent for all such particulates. Additionally, ambient general particulate matter is not solely composed of combustion source particulates. Diesel exhaust particulate demonstrates immune system effects resulting in adverse health outcomes (e.g. exacerbation of asthma and allergic rhinitis) (Diaz-Sanchez *et al*., , 2000) that are not shared by other model particulates such as carbon black and crystalline silica (van Zijverden *et al*., 2000). This suggests that diesel exhaust particulate has unique toxicological properties over and above the cardiopulmonary toxic effects of PM10.

There are uncertainties associated with estimating ambient diesel exhaust particulate concentrations from ambient elemental carbon (EC) concentrations. OEHHA, ARB and other interested parties are currently working to improve the methods used to determine ambient diesel exhaust particulate concentrations. However, the use of environmental EC measurements to extrapolate environmental diesel exhaust concentrations is generally accepted and has been used in recent published studies of the health effects of environmental diesel exhaust (Fromme *et al*., 1998; Steenland *et al*., 1998).

Finally, general population exposure was properly considered, as required by the law, as part of the available data, but was not the exclusive input into the prioritization. While there may exist some uncertainties in the precise determination of the ambient diesel exhaust particulate concentrations in California, there can be no doubt that California infants and children are substantially exposed to diesel exhaust particulate on a daily basis in urban environments.

Comment 5: Infants and children are less exposed to outdoor ambient concentrations of particulates.

It is well documented in the literature that individuals are not exposed to the same pollutant concentrations that exist in ambient, outdoor air. Recent studies by the US Environmental Protection Agency investigated the relationship between ambient concentrations of particulate matter and personal exposure in Baltimore, MD, and Fresno, CA (US EPA National Exposure Research Laboratory 2000). In these studies, individuals wore personal air sampling monitors that collected exposure information. Samples were simultaneously taken of indoor concentrations and ambient concentrations. Results from these independent studies were very similar and confirmed that average indoor concentrations are roughly 50 percent less than measured ambient concentrations. Depending on season and activity, personal exposures to PM were also generally less than outdoor ambient concentrations.

Infants and children have different activity patterns than adults and this should be evaluated. The attenuating effects of indoor environments, air filtering, and personal movements generally decrease an individual's exposure to well below ambient concentrations of air pollutants. Consequently, personal exposure levels can be 50 percent lower than measured ambient concentrations.

Recommendation 5: The activity of children and infants should be documented and exposure verified to determine if exposure is a concern.

Response: As indicated in the diesel exhaust TAC document (ARB, 1998a), OEHHA and ARB placed major emphasis on the PTEAM study conducted in Riverside (Clayton *et al*., 1993; Ozkaynak *et al*, 1994). The data were from 178 California homes so the data are relatively recent and of reasonable sample size. In addition, the researchers examined the relationship between air exchange rate, penetration factor, and indoor removal rates and provided estimates for all of these variables. This study generated a penetration factor of one for $PM_{2.5}$. Thatcher and Layton (1995) in a single, well-characterized home in California also generated a penetration factor of nearly one. A recent study by Abt *et al*. (2000) reported that for particles below 0.3 µm, between 70 and 94% of the outdoor particles penetrate indoors. This number decreases to 50% for particles up to 2 µm. In addition, the paper demonstrates that the indoor concentrations concentration of particles below 3 µm was equal to the outdoor concentration. Finally, Vette *et al*. (2001) examined only one house in Fresno. They found that the indoor concentrations were lower than those outdoor for particles <0.2 µm. However, for particles larger than this, indoor and outdoor concentrations appear similar. The observed penetration rates were in the range of 0.6 to 0.8 for particles less than 2 µm. In addition, when doors where opened, the ratio approached unity. Therefore, there is ample evidence of significant indoor exposure to PM, at the relevant particle sizes. Furthermore, when windows are open, a common occurrence in the mild California climate, there will be no significant difference between indoor and outdoor concentrations of fine particles.

Children are well documented to have greater activity levels than adults, and therefore are likely to have increased personal exposures, relative to adults, because of an enhanced personal cloud of particles. In recent surveys of the activity patterns of California children and adults (Wiley *et al*. 1991a,b), it was found that children spend an average of 124 minutes/day doing active sports, walking/hiking, or outdoor recreation, vs. only 21 minutes for adults. In personal exposure studies in the Netherlands, it has been found that, given roughly the same outdoor concentrations, children have a much higher personal PM_{10} exposure than adults (Janssen *et al*., 1997, 1998). While children's homes in these studies had a mean outdoor concentration similar to that of adults (41.5 $\mu g/m^3$ vs.

38.5 $\mu g/m^3$ for adults), children's personal exposures averaged 66.8 $\mu g/m^3$ above ambient vs. 26.9 $\mu g/m^3$ above ambient for adults. This indicates a much higher "personal cloud" for children than adults. In regressions, personal activity was one of the more important contributors to the children's extra personal exposure concentration, contributing approximately 12 $\mu g/m^3$. The children's personal exposure was also some 43 $\mu g/m^3$ higher than their time-weighted average of indoor and outdoor concentrations, indicating most of the personal vs. outdoor PM_{10} difference to be due to their personal cloud, rather than generally higher PM_{10} concentrations indoors. Thus, PM exposure of a child can be substantially higher than that for adults because of the extra PM (e.g., re-entrainment of particles) that is generated by their own increased activity levels.

Comment 6: Polycyclic Aromatic Hydrocarbon (PAHs) contained in diesel exhaust are no different that PAHs contained in gasoline, propane or natural gas and do not warrant a separate listing of diesel particulate as having a disproportionate impact on infants or children

OEHHA has listed PAHs in Tier 1 and included diesel particulate in Tier 2 because of the PAHs contained in diesel particulate. Every combustion particle contains PAHs. Gasoline contains considerably higher levels of PAHs and increased particle numbers than diesel exhaust. Singling out diesel particulate on the Tier 2 list because of the PAHs present does not accurately portray exposure to PAHs in children. This finding inaccurately, and without regard to the disproportionate impacts on infants and children, ignores gasoline particles and indoor exposure to natural gas particles as containing PAHs and duplicates the listing of PAHs in Tier 1.

Recommendation 6: Obtain data on indoor air pollutants and conduct source apportionment to determine the actual combustion sources of particulate.

Response: Internal combustion (IC) engine exhaust emissions generally do contain some amount of PAHs. However, the chemical speciation, amount and apportionment between

the exhaust physical phases (gas, semi-volatile or particulate) differs depending on the engine and fuel type. Diesel exhaust generally contains considerably greater amounts of PAHs and particulate mass than does gasoline-fueled engine exhaust. Additionally, the adverse health effects of diesel exhaust are unlikely to be only due to PAHs and particulates; diesel exhaust contains a variety of toxicants, including (but not limited to) the carcinogens benzene, 1,3-butadiene and formaldehyde. Therefore, the listing of diesel exhaust is not duplicative of the PAH listing.

OEHHA has not ignored the potential health effects of gasoline engine exhaust and indoor natural gas combustion particulate on infants and children. These materials are not listed as TACs, and are not therefore subject to identification under SB25 as specific agents having differential impacts on infants and children. However, as noted by the commenter, they may contain components, *e.g.* PAHs, identified as TACs, and also contribute to PM exposures. OEHHA would welcome any appropriate study data that could be provided on these emissions sources by the commenter or any other interested parties.

Comment 7: OEHHA finds that diesel particulate disproportionately contributes to ambient air particles ten microns or smaller (PM_{10}) that have been associated with adverse respiratory health effects in children.

Exposure to diesel particulate has not been shown to exacerbate asthma in children and infants. Since diesel exhaust is only part of the PM_{10} inventory, it is reasonable that other particles may be responsible for exacerbating asthma symptoms, such as the vapor phase of all fuels that contain PAHs.

Recommendation 7: Provide a scientific basis as to why diesel particles would be connected to increased incidences of asthma in children and infants, but other fuel particles would not.

Response: There are many published studies that indicate an association between asthma exacerbation and both PM_{10} and $PM_{2.5}$. For example, Delfino (1998), Vedal *et al*. (1998), Ostro *et al*. (1995) and Pope and Dockery (1992) all report associations between daily asthmatic symptoms in children and exposure to particulate matter. The sample sizes in these studies range from 25 to over 200. Besides these U.S. studies, there are over a dozen other studies reporting associations between short-term changes in PM and asthma from Europe and Latin America. In addition, several studies report associations between exacerbation of asthma in response to longer term exposure to particulate matter (e.g., McConnell *et al*., 1999, Zemp *et al*., 1999). These studies, conducted over a wide range of co-factors, consistently implicate PM_{10} or $PM_{2.5}$ in exacerbating asthma. Since diesel exhaust is a contributor to PM_{10} and $PM_{2.5}$, it is likely to also be associated with these adverse outcomes.

Additionally, ample mechanistic data exists indicating that diesel exhaust does exacerbate asthma and allergic rhinitis (Diaz-Sanchez *et al*., 2000). Asthma incidence is more prevalent in children than adults, suggesting that, on a population-wide basis, exacerbation of asthma by diesel exhaust will have a disproportionate impact on children. In addition, as noted in the draft document, the smaller airway diameter of children predisposes to more severe sequelae of asthma attacks. Hospitalization rates of children 0 to 4 years of age for asthma are much higher than any other age grouping (CDC, 1996a). Exhaust from other engines and/or fuel types may also exacerbate asthma in children. We agree with the comment that there are likely to be other non-diesel related particles that are associated with asthma exacerbation, but this does not render the diesel particles harmless. The lack of data for other engines and/or fuel types does not mean that diesel exhaust particulate matter should not be listed in the SB25 prioritization.